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The Treatment of Experimental *Salmonella typhi-murium* Infection in Turkey Poults and Chicks

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RECENT studies (Grumbles, Wills & Boney, 1954; Smith, 1954b, 1954c, 1955b; Cosgrove, 1954; Harwood & Stunz, 1954; Gordon & Tucker, 1955) have shown furazolidone to be of considerable value in the treatment of *Salmonella pullorum* and *Salmonella gallinarum* infection in chickens and turkeys. When this agent was administered in a concentration of 0.04 per cent. in the mash for 10 days the mortality rate in these diseases was significantly decreased and very few carriers of infection were found amongst recovered birds; many more carriers resulted when the concentration and/or the duration of treatment was reduced (Smith, 1954c, 1955b).

Although *S. pullorum* and *gallinarum* are the most important salmonellae that cause disease in chickens, the position with regard to turkeys and ducks, at least in Great Britain, is quite different; *S. typhi-murium* is by far the most economically important salmonella that infects these species. In view of this, it was decided to determine whether the general conclusions formulated as a result of the detailed studies on the furazolidone therapy of *S. pullorum* and *S. gallinarum* infection might also apply to *S. typhi-murium* infection. Turkey poults as well as chicks were used for this purpose.

Materials and Methods

Turkey poults.—One-day-old unsexed Broad-breasted Bronze turkey poults (approximate weight 60 g.) were employed.

Chicks.—These were one-day-old unsexed White Leghorn chicks (approximate weight 40 g.).

Diet.—All birds were fed solely on a mash of the following composition: white fish meal, 5 per cent.; skim milk powder, 5 per cent.; decorticated ground nut meal, 5 per cent.; dried yeast, 2 per cent.; maize meal, 11¾ per cent.; ground wheat, 20 per cent.; ground barley, 20 per cent.; ground oats, 10 per cent.; middlings, 15 per cent.; grass meal, 5 per cent.; minerals, 1¼ per cent. Furazolidone in the form of a finely ground powder was added to this mash as a pre-mix when necessary and the whole adequately mixed in an electrically-earthed mixer.

Housing.—All birds were kept in metal-sided boxes, 20 by 18 inches in area, the floor being composed entirely of ½ inch wire mesh. These boxes held 10 to 15 turkey poults or 15 to 20 chicks.

In-vitro *sensitivity tests.*—A suitable range of two-fold serial dilutions of a freshly-prepared Seitz-filtered solution of furazolidone was made in tubes containing 5 ml. of sterile nutrient broth. Each series of tubes and a control tube containing medium but no agent was then inoculated with approximately 100,000 viable *S. typhi-murium* bacteria. Nine strains of *S. typhi-murium* were tested in all. The tubes were incubated at 37° C. for 24 hours and read, the lowest concentration of furazolidone that completely inhibited bacterial multiplication as judged by visual examination being recorded as the minimal inhibitory concentration.

Method of producing infection.—A 24-hour broth culture of one strain of *S. typhi-murium*, strain 9, containing approximately 5 by 10^9 bacteria per ml. was diluted 10 times with distilled water. Alkali, 0.3 g. per ml., was then added and 0.1 ml. was deposited in the oesophagus of each poult or chick by means of a pipette, care being taken to avoid contaminating the epiglottis. The alkali employed was a powder of the following composition: powdered chalk, 40 per cent.; colloidal kaolin, 43 per cent.; magnesium trisilicate, 17 per cent.

The examination of faeces for S. typhi-murium.—Cloacal swabs taken to include a liberal portion of faeces were incubated in selenite-F medium

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(Hobbs & Allison, 1945) at 37° C. for 24 hours. Subcultures were then made on to plates of desoxycholate-citrate-agar containing 1 per cent. each of sucrose and salicin (Smith & Buxton, 1951), which were incubated at 37° C. for 24 hours and examined for the presence of *S. typhi-murium*. In some cases direct cultures of the swabs on to desoxycholate-citrate-agar were also made.

Examination of organs for S. typhi-murium.—The whole of the heart, spleen, gonads and approximately one-third of that part of the liver containing the gall bladder of each bird were removed, pooled, ground in a mortar containing sterile sand and added to 50 ml. of selenite-F medium. This material was then examined for *S. typhi-murium* in the manner previously described. When the intestinal tract was examined a portion was taken; slit longitudinally, washed well in tap water, immersed in industrial alcohol twice for half a minute and finally washed and ground in a mortar. This material was always cultured separately from that of the organs referred to above. The results also are considered separately.

The demonstration of furazolidone in intestinal contents and other material.—One to 3 ml. of material was made up to 9 ml. with phosphate buffer in a centrifuge tube, shaken well for 10 to 20 minutes and then centrifuged at 3,000 revolutions per minute for 40 minutes in an angle centrifuge. The antibiotic content of the supernatant fluid was then assayed by a cup plate technique in a manner previously described (Smith, 1954a).

Results

The in-vitro *sensitivity of strains of* S. typhi-murium *to furazolidone*

The sensitivity of the nine strains of *S. typhi-murium* to furazolidone is shown in Table I. Previous results for *S. gallinarum* and *pullorum* have been included for comparison. The growth of all the strains of *S. typhi-murium* was completely inhibited by 1 to 2 μg. per ml. of furazolidone, these strains being two to four times as resistant to this agent as was *S. pullorum*.

TABLE I

THE *In-vitro* EFFECT OF FURAZOLIDONE ON *S. typhi-murium*

Strain Number	Minimal inhibitory concentration (μg. per ml.)
2, 3, 4, 9.	1·0
1, 5, 6, 7, 8.	2·0
S. pullorum	0·25–0·5
S. gallinarum	0·5–1·0

The treatment of turkey poults infected with S. typhi-murium *with furazolidone*

The results of feeding mash containing 0.04 per cent. furazolidone to turkey poults for 10 days commencing 0, 2 and 4 days after infection with *S. typhi-murium* are shown in Table II. The examination for carriers was made five days after treatment was discontinued when the poults were killed. The mortality rate in the 0 and 2 day groups was 10 times less than in the untreated group. The effect of treatment was less apparent in the poults in which treatment was commenced four days after infection as a considerable mortality had occurred by that time; this mortality is included in the figures shown in Table II. In comparison with previous studies on the furazolidone therapy of *S. pullorum* and *gallinarum*

TABLE II

THE FURAZOLIDONE TREATMENT OF GROUPS OF 25 TURKEY POULTS INFECTED WITH *S. typhi-murium*

Commencement of treatment (days after infection)	Per cent. that died	Per cent. of survivors		
		Faecal +	Organ +	Organ + or faecal +
0	8	52	52	69
2	8	55	52	91
4	32	41	41	64
Untreated	80	100	100	100

Treatment, 0·04 per cent. furazolidone in the mash, was applied for 10 days.
+ = containing *S. typhi-murium*.

TABLE III

THE TREATMENT OF GROUPS OF 25 INFECTED CHICKS WITH DIFFERENT CONCENTRATIONS OF FURAZOLIDONE IN THE FOOD

Per cent. of furazolidone in the food	Per cent. that died	Per cent. of survivors			
		organ + faeces +	organ + faeces −	organ − faeces +	organ − faeces −
0	32	100 (82)	0 (12)	0 (6)	0 (0)
0·04	0	24 (0)	8 (4)	52 (36)	16 (64)
0·01	12	33 (0)	9 (0)	45 (55)	13 (45)
0·02	52	50 (0)	0 (0)	25 (58)	25 (32)
0·2 (not infected)	68	0 (0)	0 (0)	0 (0)	100 (100)

Treatment was commenced three days after infection and continued for 10 days.
The figures in brackets refer to the results obtained by direct culture instead of by selenite medium.

infection, the proportion of treated survivors that were carriers of infection was disappointingly high; *S. typhi-murium* was isolated from the faeces or organs of 64 to 91 per cent. of the survivors in the treated groups compared with 100 per cent. in the untreated groups.

The effect of feeding mash containing different concentrations of furazolidone on the development of carriers amongst chicks infected with S. typhi-murium

Investigations were then carried out in an attempt to discover the reason for the high proportion of carriers among the treated survivors in the previous experiment. Chicks were used for this purpose in view of their cheapness and availability.

The effect of the continuous administration of different concentrations of furazolidone in the mash for 10 days to infected chicks commencing three days after infection was first studied. The results are illustrated in Table III. One group of chicks fed mash containing 0.2 per cent. furazolidone was not infected so that they could be used as an indicator of the toxicity of the agent; 68 per cent. of this group died exhibiting nervous symptoms. None of the

chicks treated with 0.04 per cent. furazolidone died from *S. typhi-murium* infection compared with 32 per cent. of the untreated chicks. There was little difference in the carrier rate in the differently treated groups; 0.04 per cent. was as satisfactory as the higher concentrations. The carrier rate was higher in the survivors of the untreated than in the treated groups. The amount of residual infection, as evidenced by the amount of growth of *S. typhi-murium* occurring on plates of desoxycholate-citrate-agar inoculated directly from the organs and faeces, was also higher in the untreated than the treated groups. Another interesting observation was the fact that a high proportion of treated survivors were harbouring *S. typhi-murium* in their faeces but not in their organs, the figure being as high as 52 per cent. in the 0.04 per cent. groups.

The effect of administering furazolidone to chicks before infection with S. typhi-murium

When mash containing 0.04 per cent. furazolidone was fed continuously to chicks for 10 days commencing three days before infection (Table IV), 71 per cent. of the treated survivors were found to be still infected at the end of the treatment period, a high proportion of them, 42 per cent., harbouring the bacteria in their faeces but not in their organs. The low mortality rate in the untreated group was undoubtedly associated with the fact that these chicks were necessarily three days old when infected, not one day old as were the chicks in the other experiments; the resistance of chicks to fatal salmonella infection is known to increase sharply with age.

The intestinal wall of the 10 surviving chicks in the treated group that were organ negative and faeces positive were examined for the presence of *S. typhi-murium* in the manner previously described. Negative results were obtained indicating the probability that the *S. typhi-murium* found in the intestinal contents did not emanate from the intestinal wall. Since the treatment, before culture, of the intestinal wall involved the use of alcohol, it was thought possible that this might have killed any salmonellae it contained; when the intestinal tract of chicks that had died from acute *S. typhi-murium* infection was similarly examined, this was not shown to be the case.

The repeated treatment with furazolidone of chicks infected with S. typhi-murium

The effect of repeating the treatment of 0.04 per cent. furazolidone in the food for 10 days is illustrated in Table V. The first course of treatment commenced five days, and the second 22 days after infection. The mortality rate in the group that received both courses of treatment and the one that received only the first course was only one-ninth that of the untreated controls. After the first course of treatment, 63 and 67 per cent. respectively of the survivors in these two groups were excreting *S. typhi-murium* in their faeces, compared with 100 per cent. of the untreated controls. The number of carriers in all groups was much less after the second course of treatment, although, strangely enough, more carriers were present in the group that had been treated twice than in the other groups, a high proportion of the infected chicks harbouring *S. typhi-murium* in their faeces but not in their organs.

TABLE IV

THE EFFECT OF COMMENCING FURAZOLIDONE TREATMENT OF GROUPS OF 25 CHICKS THREE DAYS BEFORE INFECTION WITH *S. typhi-murium*

Treatment	Per cent. that died	Per cent. of survivors			
		organ + faeces +	organ + faeces −	organ − faeces +	organ − faeces −
0·04 per cent. in food for 10 days	4	29	0	42	29
None	8	56	4	13	22

TABLE V

THE EFFECT OF MORE THAN ONE FURAZOLIDONE TREATMENT ON GROUPS OF 25 CHICKS INFECTED WITH *S. typhi-murium*

Number of treatments	Per cent. that died	Per cent. of survivors after		
		first treatment period faecal +	second treatment period faecal +	second treatment period organ +
2	4	63	33	4
1	4	67	4	0
0	36	100	19	12

Each treatment with 0·04 per cent. furazolidone lasted for 10 days, commencing 5 and 22 days after infection.

The excretion of S. typhi-murium *in the faeces of infected chicks*

Cloacal swabs of six chicks infected orally with *S. typhi-murium* when 10 days old were examined at intervals of 12 hours for eight days after infection. It was always possible to isolate *S. typhi-murium* from each specimen examined. Three of the chicks were killed eight days after infection and the numbers of *S. typhi-murium* in various organs ascertained by the method of Miles & Misra (1938) using desoxycholate-citrate-agar. Much higher concentrations of *S. typhi-murium* were found in the intestinal contents than in either the liver, intestinal wall, bile or blood. In one chick the figure for the intestinal contents was 100,000 per gramme, the next most highly infected organ being the intestinal wall with 8,000 per gramme.

The above experiment was repeated using *S. gallinarum* instead of *S. typhi-murium*. This organism was found in the faeces six hours after infection but not again until two and a half days. After this time it was found in the faeces in small numbers only, never in such large numbers as was the case with *S. typhi-murium* nor so numerously as in the organs. This observation was in accordance with previous observations on *S. gallinarum* infection (Smith, 1955a).

The possibility of cross-infection occurring in the boxes used for experiment

Thirteen healthy day-old chicks were placed in the same box as 12 chicks infected with *S. typhi-murium*. When faecal-tested three and six days later, they were all found to have become infected.

Since this experiment indicated that cross-infection after cessation of furazolidone treatment might have possibly accounted for the high proportion of faecal excreters in the treated groups in previous experiment, the faeces of seven infected chicks receiving 0.04 per cent. furazolidone in the food were tested at seven and nine days during the treatment period. On both occasions, *S. typhi-murium* was isolated from the faeces of all seven chicks. Although high concentrations of furazolidone were demonstrated in the crop contents of these chicks after they were killed, it was never found in their faeces.

The distribution of furazolidone in the alimentary tract of chickens

The contents of the crop, gizzard, duodenum, middle portion of the ileum, caeca and rectum of six chickens fed continuously on mash containing 0.04 per cent. furazolidone for one to three days were examined for the presence of furazolidone. High concentrations were demonstrable in the crop and gizzard contents, and lower concentrations in the duodenal contents. None was found in the ileum, caeca or rectum. When the experiment was repeated using terramycin instead of furazolidone, high levels were noted in the contents of all the alimentary organs. The method of assay was approximately equally sensitive for terramycin as for furazolidone.

The survival of furazolidone in the contents of the alimentary tract

Furazolidone was added to the contents of the different portions of the alimentary tract of a healthy chicken to give a final concentration of 0.04 per cent. These were then incubated at 37° C. for two hours and assayed for furazolidone. This agent was found apparently in maximum amounts compatible with its solubility in the contents of all the organs except the rectum; smaller amounts of furazolidone were found in the contents of this organ. When the experiment was repeated using rectal contents from four different chickens a similar effect was noted. It should be pointed out in regard to these experiments that the solubility of furazolidone under ordinary laboratory conditions was not considered to be greatly in excess of 20 μg. per ml. in distilled water.

Discussion

The results indicate that furazolidone may be of considerable value in the control of natural outbreaks of *S. typhi-murium* infection in poultry. Although a high proportion of the treated chicks and poults used in the present studies remained carriers of infection, they appeared clinically healthy at the end of the treatment period and grew normally thereafter. This was not unexpected as *S. typhi-murium* infection is essentially a serious disease of young poultry only. It would be unwise to retain these birds for breeding as their progeny would probably become infected and routine treatment of all young stock in such a flock would have to be carried out.

From the public health point of view, the high proportion of carriers that result following furazolidone treatment may be of some consequence since *S. typhi-murium* is a common cause of food-poisoning in man. Infected duck eggs are frequently incriminated in this respect and, recently, there have been reports of *S. typhi-murium* in man being contracted from turkeys (Abelseth & Robertson, 1953; Pereira & Blaxland, 1955). Although it may be argued that, in outbreaks left untreated, carriers still arise, it is conceivable that they would be proportionately greater in treated flocks since many birds that would become carriers would have died had they been denied treatment. If only for these reasons, furazolidone must not be regarded as the final answer to *S. typhi-murium* infection in poultry.

The high proportion of carriers found in treated birds in these studies contrasted markedly with the results of similar studies on the furazolidone treatment of *S. pullorum* and *gallinarum* infection (Smith, 1954c, 1955b). Previous work (Smith, 1955a) had shown that *S. gallinarum* was unable to colonise the alimentary contents of chickens, any *S. gallinarum* found in the contents having originated from an infected focus in the tissues. The position with regard to *S. typhi-murium* is quite different; the present studies have shown that it can colonise the alimentary contents and may often be isolated from that site in infected chicks and not from any of the organs. *S. typhi-murium* was often found much more plentifully in the alimentary contents than in the tissues of infected chicks, often in very large numbers, indicating that it was able to multiply in the contents. This ability of *S. typhi-murium* to colonise the alimentary contents, considered with the fact that very little furazolidone could be demonstrated in the lower bowel of chicks fed mash medicated with 0.04 per cent. furazolidone, provides a reasonable explanation for the large numbers of carriers that arise after treatment.

Summary

1. Furazolidone, 0.04 per cent. in the mash fed continuously for 10 days, was very effective in controlling the mortality associated with experimental *S. typhi-murium* infection in turkey poults and chicks.

2. Even when treatment was commenced three days before infection, a high proportion of treated birds became carriers of *S. typhi-murium*. The faeces were found to be more commonly infected than were the organs.

3. Increasing the concentration of furazolidone, or repeating the treatment had no effect in decreasing the carrier rate. A concentration of 0.2 per cent. in the mash proved very toxic for chicks.

4. The reason for the high proportion of carriers amongst treated birds and their public health significance is discussed.

Acknowledgments.—I wish to express my sincere thanks to Mr. W. E. Crabb, A.I.M.L.T., and Miss G. Anderson for their capable technical help. I am also grateful to Dr. R. F. Gordon for providing the facilities for carrying out this work.

The furazolidone was kindly supplied by Messrs. A. J. White Ltd.

(Continued at foot of col. 1, page 753)

Warfarin Poisoning in a Dog

BY

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SINCE there is as yet no test for detecting Warfarin (3-α-acetonylbenzyl-4-hydroxy-coumarin) in *post-mortem* material, the diagnosis of Warfarin poisoning must rest on evidence that the animal concerned has eaten Warfarin in possibly toxic quantities, combined with the clinical features (reduced prothrombin) or the *post-mortem* findings. In the instance reported, the dog concerned is known to have eaten Warfarin and the *post-mortem* findings were compatible with a diagnosis of Warfarin poisoning.

In a public-house in the City of London, there was found indication of a severe infestation with mice and there were signs that the ship rat (*Rattus rattus*) was also present. To eliminate the infestation poisoned bait containing Warfarin to a final concentration of 0.025 per cent. ("Warfarin 5" one part, dry oatmeal 19 parts) was used, the first bait being put down on February 9th, 1955, and the last taken up on February 25th. The bait was put down in open tin lids, the owners having been told to keep their dog under control. Near the kitchen a bait of 1 oz. was taken on the first day and, as it was assumed that rats had taken it, further baits of 2 oz. were put down there on each succeeding day. The rodent operator was told on February 18th that the dog, a mongrel bitch weighing about 30 lb., had been seen eating the bait on one occasion. It was estimated that the dog could have taken at the most no more than 19 oz. of the bait. (It is generally reckoned that repeated small doses are more likely to be poisonous than a single large dose.) On February 23rd, the dog was noted to be unwell and examination at the Royal Veterinary College Clinic showed a general appearance of malaise and stiffness in the hind quarters. The temperature was normal and there was no jaundice. No estimation was made of the blood prothrombin. The dog was sent home with a guarded prognosis but died on the night of February 25th/26th.

Post-mortem examination showed the mucous membranes to be very pale. There was extensive fresh haemorrhage in the fascia of the abdominal and lumbar muscles, under the peritoneum along the mesenteric border of the large intestine, around the kidneys and in the sublumbar and pelvic region, in the subserosa and mucosa of the urinary bladder (which was empty), over the ventral half of the pericardial sac, in the mediastinum and over the anterior part of the sternum, and into the free part of the tongue. There were no obvious haemorrhages under the endocardium or epicardium, or into the alimentary canal, but the mucosa of the middle part of the trachea was diffusely reddened. The left lung showed marked hypostatic congestion. The stomach was empty. There were no obvious haemorrhages on or in the brain. The liver was pale, as were the kidneys and myocardium, and the course of the main intra-hepatic bile-ducts was indicated by yellow-green staining of the surrounding liver tissue. The blood in the vessels did not appear to have clotted.

The cause of death was a massive intra-abdominal haemorrhage for which no focal source was detected. The clot occupied much of the left part of the abdominal cavity and was of an unusual nature, in that it quivered very freely at the slightest movement of the table on which the dog was lying.

Histological examination of various tissues, besides confirming the above macroscopic observations, showed minute scattered haemorrhages in the myocardium and brain, cloudy swelling of the liver and kidney, and small subcapsular haemorrhages in the spleen. Around the portal tracts, *post-mortem* autolysis was more marked, corresponding to the bile-stained macroscopic areas. In the kidney, the glomeruli, which did not fill the distended capsular spaces, appeared more cellular and less vascular than normal; the cells had small, darkly-staining rounded single nuclei, and a moderate amount of cytoplasm. A small number of capsular spaces contained prolapsed tubular epithelium.

Summary

A case is described in which circumstantial and *post-mortem* evidence pointed to a diagnosis of Warfarin poisoning in a dog. A mongrel bitch, living on premises where Warfarin was being used, was seen to be eating the bait on one occasion, but could have taken at most no more than 19 oz. of the bait, containing Warfarin to a final concentration of 0.025 per cent. The bitch died, after showing general malaise for about two days, from massive intra-abdominal haemorrhage, and other haemorrhages were found at autopsy.

(Continued from page 752).

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Fracture of the Olecranon Process of the Ulna in the Dog

BY

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FRACTURE of the olecranon process of the ulna is quite common in the cat, and is not rare in the dog. Owing to the fact that the three heads of the triceps muscle are attached to the olecranon, and also the much less powerful tensor fasciae antibrachii, all of which exert a forward pull, there is great difficulty in obtaining satisfactory immobilisation of this fracture.

It was decided, therefore, to perform an open reduction in the case to be described, and insert a trocar-pointed Steinmann pin down through the olecranon and into the ulna shaft as described by Brinker.

The subject was a frail 18-month-old miniature poodle dog. Intravenous nembutal anaesthesia was employed, the skin incised over the centre of the dorsal and posterior aspect of the olecranon, the process, the fracture and the local anatomy were easily identified. The bone of the olecranon is the densest bone through which I have had occasion to insert a pin. Once through the olecranon the rest was easy. The result is shown in Plate I. The only other treatment was a plaster cast over the carpus to maintain this joint in extension in order to prevent as far as possible (i) tendon shortening, (ii) the dog avoiding all use of the limb. It would seem, in view of the sequel in this case, that my reasoning was at fault and some different adjunctive treatment is indicated in these cases.

The dog was re-presented in seven days for suture removal. Everything was apparently normal, and the dog was allowing the foot to touch the ground. He was next seen 14 days later, when it was obvious on palpation that the olecranon had become displaced forward. Radiography (Plate II) revealed that the pin had become bent forward. The operation was repeated, the bent pin removed, a fresh one inserted, and no carpal extension attempted.

This case is reported because (i) these pins are very strong, and it is impossible to bend a piece just over two inches long with the hand in the absence of suitable tools, (ii) it would seem that the only possible way to repair this fracture satisfactorily may be defeated by the apparently tremendous force exerted by the triceps in the dog.

It is of great interest to note the further operation carried out on this dog. The bent pin was removed, a straight pin the same size and length as the previous one was inserted into the identical drilled holes, no attempt was made to plaster or otherwise interfere with the dog's use of the limb, and after six weeks normal function was restored. It is to be noted that by the time the second pin was inserted there was marked atrophy of the muscles of the whole foreleg.

A Case of Heavy Whipworm Infestation in a Dog and its Successful Treatment

BY

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Norwich

HEAVY infestation of the dog with the whipworm, *Trichuris vulpis*, is comparatively rare in this country, although very frequent in the North American continent. Whipworms inhabit the caecum and colon of the dog and produce a variety of symptoms including diarrhoea, anaemia, unthriftiness and dermatitis.

Until recently a safe and effective agent to remove the whipworms from the dog has not been available, although many different drugs have been tried for this purpose including santonin, nicotine, oil of chenopodium, tetrachlorethylene, hexylresorcinol and hydrogen peroxide. So unsuccessful has medical treatment been that in chronic cases, surgical removal of the infected caecum has been resorted to in an effort to effect an improvement in the host's condition.

Recently a new trichuricidal agent, phthalofyne, (3-methyl-1-pentyn-3-yl-acid phthalate), has come to the fore in America as a safe and effective oral treatment for the removal of this troublesome parasite. A supply of this drug was obtained from Messrs. Pitman-Moore Co. of Indianopolis, Ind. U.S.A., for trial in the case described below.

Subject. Two-year-old Springer Spaniel bitch.

History. The present owner purchased the bitch when she was eight months old. Just prior to his collecting her she had what was described as a mild attack of a distemper-like disease. Since she came into the owner's possession she has had a persistent mucoid type of diarrhoea, the faeces never attaining normal consistency.

At examination the bitch was rather emaciated but apparently eating well and full of energy. The coat was very dry and full of scurf and appeared to cause a lot of irritation. The owner also reported a lot of flatulence, which would seem to have been aggravated by a wind-sucking habit the bitch had developed when she cleaned herself.

Successive faeces samples showed the presence of large numbers of eggs of *Trichuris* spp.

Treatment.—Following the advice of Mönnig, a seven-day course of treatment with a daily dose of santonin ½ gr. and calomel ½ gr. was tried on two occasions without any apparent effect on the clinical condition of the bitch or the faecal worm egg count.

Before treatment with "phthalofyne," a faecal worm egg count showed the presence of 18,650 e.p.g.

The bitch was given three tablets, each containing 0.912 g. of the drug, after a preliminary 24-hour fasting period. No vomiting occurred and rather surprisingly the animal did not defaecate for 78 hours after dosing. A faeces sample at that time showed dozens of whipworms, all rather badly macerated. Another faeces sample taken a week after dosing showed that the egg count had dropped to 200 e.p.g. A repeat dose was given seven weeks later and a post-dosing sample showed no Trichuris eggs.

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KNIGHT, R. W. j., & PITTAWAY, E. M.—FRACTURE OF THE OLECRANON PROCESS OF THE ULNA IN THE DOG

PLATE 1.

PLATE 2.

Cobalt Therapy in Farm Practice with Special Reference to Hill Farms

BY
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ALTHOUGH the syndrome of cobalt deficiency in young stock is now well-known and its treatment well established there are still some difficulties in preventing the condition under a few different methods of farm practice. For arable grassland and for low-ground permanent pastures a dressing of 2 lb. cobalt sulphate per acre mixed with a suitable filler, such as superphosphates, will raise cobalt-deficient pasture to a level of adequacy, and keep it adequate for three or four years, according to the soil type and grass seeds mixture. On hill land dressing the herbage with cobalt salts is a more difficult practice. Nevertheless by spreading the cobalt on a percentage of their grazings, but allowing their whole flock access to the dressed area for a period each day, many hill farmers have successfully prevented cobalt deficiency in their lambs. Many hill farms, however, especially in the Highlands of Scotland, have no grazings which could be dressed by machine, and spreading cobalt salts over 50 to 100 acres by hand is laborious and very often would be most uneconomic when the extent of the grazings and the stocking rate are considered. An alternative method of preventing cobalt deficiency is to feed mineral mixtures supplemented with cobalt, and this has proved most efficacious on many hillsides where the flocks are skilfully shepherded and made to pass the troughs containing the mineral mixtures each day. Unfortunately, many sheep will not eat mineral mixtures even after being attracted to the troughs by a ration of bruised oats or other feeding stuff. Much field work has been carried out to make mineral mixtures more attractive to sheep by the addition of substances which altered the taste, smell and colour of the mixtures, but to date we have been unable to ascertain what perfume a sheep prefers, what taste tickles its palate, or what colour attracts its eye.

Therefore the problem of cobalt therapy on many hill farms where the soil is cobalt-deficient and the topography such that dressing the herbage with cobalt salts is extremely difficult, and in many cases impossible, has still to be solved. It is important that any method which is advised must be workable under the existing farm routine and at the same time give a satisfactory financial return.

Individual dosing of lambs with cobalt salts has often been adopted as a curative procedure in the case of lambs showing clinical symptoms of cobalt deficiency. The lambs and their mothers have to be folded near the farm building to be of ready access as the dosing must be carried out at least once per week to obtain any marked degree of recovery. Weekly gatherings of lambs and weekly dosing with cobalt salts cannot be regarded as a practical method for the prevention of cobalt deficiency on a hill farm. The labour of gathering and shedding the lambs could not be contemplated as part of a routine measure unless the usual sheep gatherings were utilised.[1] On most hill farms gatherings take place at approximately monthly intervals throughout the first six months of a lamb's life, and thus it would be possible to use such gatherings for individual dosing of the flock provided that monthly dosing with cobalt salts would prevent the condition.

In a series of experiments we have found that in lambs grazing cobalt deficient herbage liveweight gain is approximately inversely proportional to the interval between dosing provided equivalent doses of cobalt salts are given. The summary of the results of several such experiments are shown in Table I (*overleaf*).

From Table I it can be observed that although grazing cobalt-dressed pasture produced the highest liveweight gains yet dosing daily with 1 mg. cobalt or weekly with 7 mg. cobalt gave reasonably good

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Since treatment, all traces of the diarrhoea have disappeared and the animal's bowel actions are normal for the first time. Her coat and general condition have improved and the skin is much less irritable.

Summary.—Successful treatment of a case of heavy whipworm (*Trichuris vulpis*), infestation in a Springer Spaniel bitch using a new trichuricidal agent, phthalofyne, is recorded.

Acknowledgments.—The writer is indebted to J. E. Sloan, Esq., B.SC., of the Cooper Research Station, Berkhamsted, for examining the faeces samples and for suggesting the use of phthalofyne,* and to Messrs. Pitman-Moore Co. for a supply of the drug.

Reference

MÖNNIG, H. O. (1941). "Veterinary Helminthology and Entomology." Baillière, Tindall & Cox, London. 2nd Edition, p. 152.

*Phthalofyne is marketed under the trade mark of "Whipcide" by Messrs. Pitman-Moore Co.

TABLE I
VARIATION IN LIVE-WEIGHT GAIN ACCORDING TO INTERVAL BETWEEN DOSING

Dose	Number of animals	Average live-weight gain June 1st to August 31st	Standard error of average
		lb.	
(*a*) Lambs grazing cobalt-deficient pasture	200	26·6	± 1·38
(*b*) As (*a*) but receiving 1 mg. cobalt per day	50	55·6	± 0·59
(*c*) As (*a*) but receiving 7 mg. cobalt per 7 days	50	52·4	± 0·39
(*d*) As (*a*) but receiving 14 mg. cobalt per 14 days	50	39·4	± 0·62
(*e*) As (*a*) but receiving 28 mg. cobalt per 28 days	50	30·1	± 0·79
(*f*) Lambs grazing cobalt-dressed pasture	100	57·2	± 0·92

results. When dosing was carried out at fortnightly and monthly intervals with 14 mg. and 28 mg. respectively, the liveweight gains were very small and of the same order as those of lambs grazing the cobalt-deficient pasture and given no cobalt supplement, but yet the difference between fortnightly and monthly dosing was apparent. The difference between weekly and fortnightly dosing was very marked. These results can be explained if we assume that the cobalt is utilised by the ruminant in the rumen and that cobalt given weekly allows the rumen to function normally, whereas if given fortnightly, or at longer intervals, there is too long a period of cobalt deficiency in the rumen to allow of the rumen processes being exercised to a normal extent.

In the light of the above results and the above hypothesis further experiments were carried out to ascertain if massive doses of a cobalt salt at monthly intervals would allow of liveweight gains comparable to those obtained by weekly or daily dosing of the usual therapeutic dose of 1 mg. cobalt per day.

On a farm in Kirkcudbrightshire previous experiments (Stewart, Mitchell, Stewart & Young, 1946) had demonstrated that the pasture was cobalt-deficient and that dressings of cobalt sulphate at the rate of 2 lb. per acre would increase the cobalt content of the pasture from 0.09 p.p.m. cobalt to 0.38 p.p.m. cobalt and that the liveweight gain of lambs grazing the cobalt-dressed pasture was over 20 lb. greater in 15 weeks than that of lambs grazing the cobalt-deficient pasture.

The field, 22 acres in area, had been divided into two equal parts by an impassable wire fence. One half of the field had received a top-dressing of superphosphates at the rate of 1½ cwt. per acre. The other half had also been top-dressed with superphosphates at the rate of 1½ cwt. per acre but 2 lb. of cobalt sulphate was mixed with each 1½ cwt. of superphosphate applied. Thus, at the beginning of the present experiment the cobalt content of the pasture of the half which had received no cobalt dressing was approximately 0.09 p.p.m. cobalt and that of the half which had received the cobalt dressing approximately 0.40 p.p.m. On June 5th, 1952, 19 lambs were placed on the cobalt-dressed half of the field and 19 on the other half. Thirteen of the lambs on the cobalt-deficient half were given 250 mg. cobalt on June 5th, July 6th and August 3rd, that is, at approximately monthly intervals from six weeks of age. The remaining six on the cobalt-deficient pasture acted as controls. All the lambs on both sides of the fence were weighed on the days on which the lambs received their dose of cobalt. Weighings were also carrried out on August 24th and September 21st.

The total liveweight and liveweight increases at the various weighings are shown in Table II.

At August 3rd all six control lambs showed typical symptoms of cobalt deficiency and were given 250 mg. cobalt on that date. The smallest of the six died on August 6th and therefore the values for the control group of August 24th and September 21st are the averages of the five remaining lambs. Also it must be borne in mind that these final averages would have been much lower but for the cobalt dosing of August 3rd. Evidence of this is seen in the control group showing an average gain of 7.63 lb. during August when during July the average liveweight gain was only 2.84 lb.

TABLE II
EFFECT OF MONTHLY DOSING OF 250 MG. COBALT ON LIVE-WEIGHT INCREASE OF LAMBS—1952 EXPERIMENT

Date of weighing	Lambs grazing cobalt-dressed pasture		Lambs grazing cobalt-deficient pasture			
			Group receiving 250 mg. cobalt monthly		Control group	
	Average weight of lambs	Average increase in live-weight	Average weight of lambs	Average increase in live-weight	Average weight of lambs	Average increase in live-weight
	lb.	lb.	lb.	lb.	lb.	lb.
June 1st, 1952	30·79	—	32·77	—	32·33	—
July 6th, 1952	52·58	21·79	51·62	18·85	44·33	12·00
August 3rd, 1952	66·10	13·52	65·61	13·99	47·17	2·84
August 24th, 1952	73·74	7·64	69·08	3·47	54·80	7·63
September 21st, 1952	83·42	9·68	79·83	10·75	63·80	9·00
Average increase in weight during experiment		52·63		47·06		31·47
Standard error of average increase in live-weight		± 2·26		± 0·55		± 0·39

(*Continued on page 757*)

TABLE III

EFFECT OF MONTHLY DOSING OF 250 MG. COBALT ON LIVE-WEIGHT INCREASE OF LAMBS—1953 EXPERIMENT

Date of weighing	Lambs grazing cobalt-dressed pasture		Lambs grazing cobalt-deficient pasture			
			Group receiving 250 mg. cobalt monthly		Control group	
	Average weight of lambs	Average increase in live-weight	Average weight of lambs	Average increase in live-weight	Average weight of lambs	Average increase in live-weight
	lb.	lb.	lb.	lb.	lb.	lb.
June 7th, 1953	39·50	—	34·20	—	37·90	—
July 5th, 1953	57·80	18·30	49·90	15·70	51·70	13·80
August 2nd, 1953	72·25	14·45	62·90	13·00	62·90	11·20
August 30th, 1953	82·35	10·10	73·30	10·40	71·10	8·20
September 26th, 1953	89·15	6·80	80·80	7·50	72·00	0·90
Average increase in weight during experiment		49·65		46·60		34·10
Standard error of average increase in live-weight		± 0·68		± 0·93		± 1·21

There was a marked difference in liveweight increase between the lambs dosed monthly with 250 mg. Co and the control lambs—15 lb. in favour of the cobalt-dosed group. The difference in liveweight increase between the cobalt-dosed group and the group grazing the cobalt-dressed pasture was 5.6 lb. Therefore, although the dosed group made a substantial live-weight gain as compared with the control group receiving no cobalt, this liveweight gain was less than that of the lambs obtaining their adequate daily dose of cobalt from the cobalt-enriched herbage on the other side of the fence. The liveweight gain of the monthly dosed group was of the same order of live-weight gain as those in the top section of Table I.

The experiment was repeated in 1953, the only difference in procedure being that of the lambs on the cobalt-deficient half of the field 10 were dosed monthly with 250 mg. cobalt and 10 acted as controls receiving no cobalt supplement. On August 30th five of the 10 control lambs received 250 mg. cobalt in order to ascertain the therapeutic value of one such dose on cobalt-deficient lambs. The results are shown in Table III but the values given for the control lambs for September 26th are the averages of the five lambs which had remained undosed throughout the experiment.

The results shown in Table III amply confirm those of 1952. The lambs which had received the monthly dose of cobalt had a liveweight increase on September 26th of 12.5 lb. greater than the controls, but 3 lb. lower than those grazing the cobalt-dressed pasture. Whereas the group grazing the cobalt-dressed pasture during September gained 6.8 lb. per head, and those dosed with cobalt monthly 7.5 lb. per head, the control lambs gained only 0.9 lb. per head. Of the five control lambs which received 250 mg. cobalt on August 30th the one which showed the worst symptoms of pine died, but the other four made a live-weight gain of 6 lb. per head during September. Thus the dose of 250 mg. cobalt given on August 30th produced a significant curative response in the control lambs.

The very similar results of the experiments carried out in two successive years show that monthly dosing with 250 mg. cobalt of lambs on cobalt-deficient pasture is a most useful method of preventing the condition of cobalt deficiency and producing healthy lambs. Although the liveweight gains of the lambs receiving the heavy monthly dose of cobalt were not so great as those of lambs receiving the cobalt daily through cobalt-rich pasture, a very important point was that none of the lambs in the monthly dosed groups of both years showed any symptoms of cobalt deficiency and appeared in excellent health and the prevention of a large number of poor ailing lambs in a flock in August and September is of great benefit to the flock-master even if these lambs are not all in the highest class.

It is possible that even larger doses of cobalt salts could be safely given monthly with even better results, although several authors have reported the occurrence of polycythemia after massive doses of cobalt. The authors, however, suggest that even the dose of 250 mg. cobalt (*i.e.*, equivalent to 1 g. cobalt chloride) is to be recommended as a means of counteracting cobalt deficiency in cobalt-deficient hill farms where other means of prevention are very difficult to carry out. As has been pointed out above, monthly dosing of lambs means dosing each lamb at every flock gathering during the summer and adds very little labour to the usual routine of a hill sheep farm. The cost of 250 mg. of cobalt is approximately one halfpenny so that the four dosings during June, July, August and September would cost twopence per lamb, from which outlay the farmer would gain a substantial benefit.

Summary

Field experiments carried out in two successive years showed that monthly dosing with 250 mg. cobalt of lambs grazing cobalt-deficient pasture is a most useful method of preventing the condition of cobalt deficiency and producing healthy lambs.

The above work was carried out under the auspices of the Hill Farm Research Committee of the Department of Agriculture for Scotland as part of their programme to increase production on hill farms.

Reference

STEWART, JAMES, MITCHELL, R. L., STEWART, A. B., & YOUNG, H. (1946). *Emp. J. Exp. Agric.*, **14**. 145.

ANNUAL POULTRY CONFERENCE

Veterinarians were again prominent in the third day of the Harper Adams Poultry Conference, the opening of which was reported in our last issue, and "The Poultry Stock Improvement Plan" was the theme for discussion. Mr. John Reid, M.R.C.V.S., whose paper in his unavoidable absence was read by Mr. G. Laugier, M.R.C.V.S., presented an excellent review of the progress made in the veterinary approach to stock improvement. After outlining the power which the Ministry employ to control poultry disease under section 46 of the Diseases of Animals Act 1950, including the specific Orders to deal with fowl pest, ornithosis and duck virus hepatitis, Mr. Reid surveyed the present health position in those flocks included in the scheme. Since 1943 the percentage of reactors to *pullorum* disease has decreased consistently to reach in 1954 a record low level. Over three and a half million birds were tested in 3,800 flocks with 0.14 reactors, less than three fowls in every 2,000 tested. About 65 per cent. of all the breeding fowl in England and Wales are in the P.S.I.P. and of about 60 million chicks distributed annually, over 70 per cent, are produced within the plan. Mr. Reid went on to point out, however, that changes in management, particularly deep litter, were resulting in the spread of *pullorum* infection in adult fowls and leading to breakdowns in control. Mr. Reid also dealt with the possible control of fowl typhoid and salmonellosis, drawing attention to the use of therapeutic agents such as furazolidone and to improvements in formaldehyde fumigation.

In reviewing the year's progress in the control of fowl pest, Mr. Reid stated that "the records show a very great improvement in 1955. The clean area policy has shown a remarkably good result, and the counties declared clean have broadly remained clean. The disease is now mainly restricted to the Eastern and South-eastern counties. There has also been a notable improvement on the part of owners in reporting suspected cases, *e.g.* in 1953, only 25 per cent. were reported; in 1954, 75 per cent. and in 1955 to date 83 per cent. However, the greatest worry is the frequency with which primary cases are linked to recent visits to breeding farms by poulterers and dealers."

The discussion was opened by Dr. Gordon of the Animal Health Trust who said that, with all due respect to the work of the N.A.A.S., the fact that the number of flocks within the P.S.I.P. had grown from less than 100 when he was first associated with the scheme in 1933, to nearly 4,000 flocks in 1955 was in itself testimony to the efficiency and enthusiasm of the Animal Health Division. "Nevertheless," said Dr. Gordon, "we must not rest on the laurels of progress in the eradication of *pullorum* disease and fowl pest"; there are too many poultry farmers who give a lip service only to routine *pullorum* testing but who do not use the veterinary diagnostic services as outlined in the plan. There are a number of diseases, fowl typhoid, salmonellosis, fowl paralysis, leucosis, etc., which are now of greater economic importance than *pullorum* disease, but for which clear cut diagnostic tests do not exist, and therefore progress in their control can only be made by close collaboration between the breeder, the veterinary advisory staff and the laboratories. Dr. Gordon outlined recent advances in the prevention and control of *salmonella* infections and fowl typhoid, both by therapeutic and hygienic measures, such as the use of furazolidone (including the control of *pullorum* carriers), germicidal egg dips and the use of aerosols in incubator hygiene. He also drew attention to recent views on the classification of the leucosis complex on a pathological basis and the implications of these views on the aetiology and transmission of the disease, particularly as they affected the breeder and his relations with his customer.

Dr. Gordon stated that although advances had been made in the control of disease on the breeding farms, little or no improvement had occurred in the control of preventable diseases on the commercial farms of this country. He felt that although national conferences of this type played a useful part, it was mainly a case of "preaching to the already converted" and that there was a great need for improvement in the advisory services' methods of disseminating information on a more local and less formal basis, so as to reach the general farmer who after all was responsible for the major part of our poultry production.

The remainder of this session was devoted to a most stimulating paper by Mr. G. E. Mann, M.A., of the N.A.A.S. on recent advances in poultry genetics. Mr. Mann pointed out that many British breeders were deterred from progeny testing by the expense in recording under present-day economics, but were apparently loathe to accept the now well-founded fact that part-time records, such as three-day trap nesting or five-day recording over a limited period, was an accurate method of selection. It was his opinion that the days of the small breeder were numbered and that the greatest progress would be made by the large breeding organisations or by a number of small pedigree breeders amalgamating. He foresaw an increase in popularity of hybrid stocks during the coming year and that this product was well worth the small premium charged for it.

The chairman of the session on breeding was Sir John Barlow, M.P., and on disease, Mr. Readman, a well-known accredited breeder from Yorkshire.

IMPORT OF ANIMAL SEMEN

The Minister of Agriculture, Fisheries and Food, and the Secretary of State for Scotland have made an Order under the Diseases of Animals Act prohibiting, except under licence, the import of semen of swine, sheep, goats, other ruminating animals (except cattle), canines, felines and poultry. The import of cattle and horse semen is already controlled by Orders made under the Agriculture (Miscellaneous Provisions) Act, 1943.

Copies of the Order, The Importation of Animal Semen Order, 1955 (S.I. 1955 No. 1390), which came into operation on September 17th, are obtainable from H.M. Stationery Office.

Current Literature

ABSTRACT

Order and Disorder in Pulmonary Function. ARNOTT, W. M. (1955). Professor of Medicine, University of Birmingham. *Brit. med. J.*, July 30th, 1955. 279–284 and August 6th, 1955. 342–348.

This long and exceedingly interesting paper, comprising two Oliver-Sharpey lectures delivered to the Royal College of Physicians, London, commences by pointing out that in the last 10 or 20 years more and more clinicians are turning to the problem of respiratory function in man and are using physiological methods both of thought and of observation in lung disease.

In the introduction the functional description devised by Richard Riley is quoted. "The lung is an organ into which inspired gas and mixed venous blood are pumped, in which the gas and blood are distributed to the alveoli where, by diffusion, equilibrium with respect to carbon dioxide and oxygen is approached and out of which expired gas and arterial blood pour fourth."

The Alveolo-capillary membrane

The alveolo-capillary membrane forms the air-blood interface at the alveolar capillaries. The diffusion capacity is the amount of oxygen that diffuses into the blood for each 1 mm. of Hg pressure difference. It is calculated from a formula based on physiological measurements of the amount of oxygen diffusing in relation to the mean pressure gradient between the alveolar gas and the alveolar capillary blood. The diffusion capacity is a function of the area of the blood-gas interface and the thickness of the intervening membrane. This thickness comprises the total distance between the inner surface of the alveolar wall and the point in the haemoglobin molecule where oxygen enters into chemical union. This diffusion distance is of the order of 2–4/μ, and in healthy lung is at its minimal value. Any pathological change with the possible exception of emphysema is likely to increase rather than diminish the distance. The rate of diffusion of a gas through tissue is directly proportional to its solubility and inversely proportional to the square root of its molecular weight. The molecular weight factor may be ignored but the solubility factor is of great practical importance. Carbon dioxide is about 25 times more soluble than oxygen and therefore it diffuses approximately that number of times more easily. Cardon dioxide can be relied on to diffuse adequately despite any increase in the diffusion distance possible within the limits of viability.

Carbon dioxide retention in the body, as in emphysema, is not the result of impaired diffusion but is due to defective alveolar ventilation.

Measurement of Diffusion Capacity

The measurements necessary to determine the pulmonary diffusion constant are not such that they can be applied by a clinical biochemist as for instance in the determination of blood urea. By rather elaborate methods it has been possible to determine that it is most likely the chief mechanism for the augmentation of the diffusion capacity is the opening up of new capillaries with perhaps a slight diminution of the diffusion distance by lung stretching, and not the dilatation of capillaries already open, as that would result in the axial stream of blood suffering the disability of an increased diffusion distance. While the requirements of respiratory function in terms of O_2 absorbed and CO_2 excreted are precisely set by the metabolic activity of the body there exists scope for quite wide fluctuations of respiratory exchange ratio as regards short intervals of time and space, in that some alveoli may be well perfused and poorly ventilated, while in others the reverse state prevails. This means that distribution is the fourth factor which along with ventilation, diffusion and circulation are the major physiological influences affecting the partial pressures of O_2 and CO_2 in gas and blood. Distribution is a factor applicable to both blood and gas. A relative deficiency of blood perfusion as opposed to gas ventilation raises the amount of CO_2 excreted relative to the oxygen absorbed, resulting in an exchange ratio above that determined by the currently prevailing metabolic state. The theoretical extreme of this distortion is a ventilated but avascular alveolus which is merely an addition to the anatomical respiratory dead space. The reverse state of affairs, where an alveolus is well perfused but has impaired ventilation greatly reduces the CO_2 excretion.

The term "pulmonary fibrosis" has been revived by the pulmonary physiologist and has been given, rather arbitrarily, new and quite rigorous functional criteria. The basic disorder which characterises this pulmonary fibrosis is an increased alveolar diffusion gradient for O_2 due principally to increase in diffusion distance and also a reduction of the blood-gas interface. It is apt to occur in diseases which produce throughout much of both lungs an increase of tissue either by cellular proliferation or by infiltration, the swelling by inhibition, of connective tissue elements, or the accumulation of fluid in connective tissue layers, or adhering by surface tension to the alveolar walls. There is by implication an element of chronicity in the concept, as the term is not applied to comparatively acute syndromes of pulmonary dysfunction of the impaired diffusion type such as occurs in pulmonary oedema. The aetiological basis of the fibrosis syndrome includes a variety of quite unrelated processes.

It is only in severe cases that the arterial blood is unsaturated with oxygen at rest, but in all but the mildest it becomes unsaturated on exercise. There is little or no CO_2 retention. The other changes are either in the nature of adaptation or due to the pathological changes involving tissues other than the alveolar capillary membrane. The increased tissue thickness of the pulmonary septa present a quite abnormal resistance to distension so that the total lung capacity, vital capacity and inspiratory reserve capacity are all much reduced. Mainly because the airways remain unimpeded the maximum ventilatory capacity is not materially reduced but there is constant hyperventilation that is achieved by an increase in frequency rather than an increase in amplitude of respiration.

Ventilatory Dysfunction

The outstanding physiological defect that is present in disease processes which partially occlude air passages or which, as in emphysema, are accompanied by gross

reduction in lung elasticity is reduction of alveolar ventilation. The result is an abnormally low alveolar oxygen tension and high CO_2 tension leading to arterial unsaturation and CO_2 retention—that is anoxaemia and hypercarbaemia.

Pulmonary Circulation

There are several broad structural and functional features of the pulmonary circulation which must be emphasised. The flow through this circuit is the output of the right ventricle and the input of the left auricle and therefore the output of the left ventricle. The balance between systemic and pulmonary flow must clearly be extremely accurate. A discrepancy of 1 ml. per beat (an error of only about 1·5 per cent.) in the same direction maintained for only a quarter of an hour would result in something like one litre increase of blood volume in one circuit at the expense of the other—a general trend that would be incompatible with life.

Another limiting circumstance is that unlike systemic capillaries, there is no call for any considerable transudation from the pulmonary capillaries. So long as there is a film of water in the alveolar walls no fluid need escape. Even that film of water represents only a trifling amount, evaporating little, as the air is probably saturated by the time it reaches the alveoli as the result of ample secretion of water from the walls of the bronchial and bronchiolar mucosa. The normal lungs have a marked capacity to absorb water, hypotonic and normal saline and other electrolyte solutions if they can be introduced in a fine spray, often called loosely an "aerosol." An anatomical feature of importance is that the pulmonary capillaries constitute a vertically unsupported network in the alveolar walls. This is quite unlike the systemic capillaries which are surrounded by a connective-tissue network, the intercellular interstices of which are filled with water, the whole providing support.

Bronchial Circulation

The bronchial circulation is capable of exerting profound influence on pressure-flow relationships in the pulmonary circuit. In diseased states, such as chronic inflammation, collapse, emphysema, etc. the evaluation of pulmonary circulatory haemodynamics becomes exceedingly complex. There is definite physiological evidence that blood can pass from artery to vein without adequate exposure to alveolar gas, this being known as an arterio-venous shunt.

Control of Pulmonary Circulation

For long there has been a controversy as to whether there is nervous control of the pulmonary circulation. Many believe that the pulmonary circulation reflects passively the cardiac activity and the systemic influences altering the distribution of blood between the two circuits.

Other workers have concluded from experimental observations that while the vaso-motor control of the lung vasculature was poorly developed and feeble as compared with the highly developed neurogenic control of the systemic circulation, its effects were functionally demonstrable as independent of changes in the systemic circuit.

It is difficult to attempt any sort of critical evaluation of the influence of hormones, drugs and "toxins," but there is evidence that the pulmonary circulation does respond to a variety of biological agents and their antagonists. The response tends to be slight in healthy states but may be large in diseased states. There are variations in the response according to the immediate environment and the recent experience of the pulmonary circulation. The indications are that the pulmonary circuit can respond in itself and that the reactions are not secondary to the systemic circuit changes.

An important recent discovery is that of the effect of gas tension on the pulmonary circulation. It has been shown that anoxia or raised inspired CO_2 tension produced an increase of pulmonary arterial pressure.

G. F. B.

BOOK REVIEW

The Calf: Its Management, Feeding and Health.

By J. H. B. ROY, M.A., DIP. AGRIC. (CANTAB.). Farmer and Stock-Breeder Publications Ltd., Dorset House, Stamford Street, London, S.E.1. Price 5s. net. 79 pages. 25 photographs and diagrams.

This excellent little booklet on the calf is extremely well produced, illustrated and printed. It brings within a limited orbit all the information about calves and calf rearing for which one has to seek so far in the rather nebulous and widely distributed literature on animal management.

PART 1—entitled MANAGEMENT AND FEEDING—covers information about parturition and the care of the calf up to the fourth day of life. There are also detailed instructions about feeding requirements and suitable systems of management up to six months of age. Reference is made to all the practical points for which one frequently seeks authentic advice, such as the quantity of milk required, number of feeds per day, temperature, encouraging calves to drink, the use of antibiotics, the importance of dry food and the time to introduce the feed, etc. There are also some helpful remarks and good diagrams of calf pens and houses. In the latter respect it is pleasing to note that the author favours the use of independent calf pens, completely separated from each other by suitable solid partitions, though he does indicate that if all goes well the open tubular type of calf pens give good results and have certain advantages.

Calves, like all young animals, dislike an overhead draught and every effort should be made to provide comfortable quarters for the most satisfactory growth rate and development.

PART 2—entitled CALF DISEASES—is a section which perhaps many veterinary surgeons may have preferred not to see, but such information as is given is common knowledge and is highly useful; and one cannot but agree that such a section makes the book more complete and satisfying. In this section, however, it is perhaps unnecessary to include parasitic gastroenteritis—a condition which can hardly be regarded as a problem in calves, at least in this country.

This is a booklet the outlay for which will be well repaid by anyone interested in calf husbandry.

News and Comment

SUPPLEMENT TO "THE VETERINARY RECORD"

A Supplement to THE VETERINARY RECORD will be issued at the end of this month and Hon. Secretaries of Divisions, B.V.A., are asked to send in their minutes for publication in this number by Monday, October 10th.

CONFERENCE ON "THE FEEDING OF FARM ANIMALS FOR HEALTH AND PRODUCTION"

With last week's issue of THE VETERINARY RECORD, the proceedings of this Conference were sent out in booklet form to every member of the Association. There has been considerable and unavoidable delay in the publication of these proceedings, but it is hoped they will be of interest to members now that they are available.

Since the distribution of the booklets last week, a few copies which have slipped from the envelopes have been returned by the Post Office; any members, therefore, who have not received their copies are asked to inform the General Secretary, B.V.A., 7, Mansfield Street, Portland Place, London, W.1, who will be pleased to replace the missing copies.

ERADICATION PLANS FOR TUBERCULOSIS

The Ministry of Agriculture, in a joint announcement with the Agricultural Departments in Great Britain, has stated that as from October 1st, 1955, the following Attested Area Orders will come into operation:—

(i) *The Tuberculosis (South-West and Mid-Wales Attested Area) Order, 1955.*

This declares an Attested Area which combines the existing Attested Area, comprising the counties of Pembroke, Cardigan, Carmarthen, and West Glamorgan, with the adjoining counties of Brecon, Radnor, Montgomery and Merioneth that were declared an Eradication Area on March 1st, 1955.

(ii) *The Tuberculosis (North-West England Attested Area) Order, 1955.*

This declares an Attested Area for Cumberland, Westmorland and parts of Durham, Lancashire and the North and West Ridings of Yorkshire that were declared an Eradication Area on March 1st, 1955.

(iii) *The Tuberculosis (Argyll and Hebrides, Central, Forth and South-West Scotland Attested Area) Order, 1955.*

This order combines the existing Attested Area, comprising the counties of Ayr, Wigtown, Kirkcudbright, Dumfries, Renfrew, Bute, Dunbarton, Stirling, Lanark, Peebles and part of Argyll, with the adjoining counties of West Lothian, Midlothian, Selkirk, Fife, Clackmannan, Kinross, part of Perth, the remainder of Argyll, part of the mainland of Inverness, together with the Outer and Inner Hebrides, that were declared Eradication Areas on March 1st, 1955.

The Minister of Agriculture, Fisheries and Food is responsible for the first two orders, and the Secretary of State for Scotland has made the third. In order to facilitate the movement of stock across the border in either direction between the separate Attested Areas in South-west Scotland and North-west England, an Amendment Order has been made by the Minister and the Secretary of State acting jointly, exempting from the licensing provisions of the Tuberculosis (Area Eradication) Order, 1950, movements of stock from one Attested Area to another where the two areas are contiguous.

Compulsory tuberculin testing has been carried out in the four Eradication Areas—certain counties of North-west England, Mid-Wales, and in Scotland, the Forth, Argyll and the Hebrides—in respect of herds not attested on March 1st, 1955, and reactors have been removed and slaughtered. Altogether some 385 herds (8,500 cattle) have been dealt with; the incidence of reactors at the first round of compulsory tuberculin tests was 14 per cent., but at the second round it was only 2 per cent. For all practical purposes, therefore, bovine tuberculosis is now non-existent in these Eradication Areas. Separate Attested Area Orders, however, are in force for the Shetland Isles and the Isles of Scilly.

The Ministry report that at June 30th, 1955, there were about 5.3 million cattle in attested herds in Great Britain, as against 2.25 million in October, 1950; an increase of 135 per cent. in under five years. Over half the cattle in the country are now in attested herds; in England 46 per cent., in Wales 76 per cent., and in Scotland 74 per cent. Approximately 1,880,000 cattle, or one-fifth of the total in Great Britain, will be in Attested Areas as at October 1st, 1955. At the present rate of progress it is hoped that the country will be free of bovine tuberculosis in about the next ten years.

Copies of the various Orders are obtainable from H.M. Stationery Office, Kingsway, London, W.C.2., or from any bookseller, price 2d. net.

SURPLUS CHICK DESTRUCTION

A pamphlet dealing with the humane destruction of surplus chicks, a problem which inevitably arises from time to time in commercial chick production, has been issued by the Animal Health Trust.

Collaborating with the Chick Producers' Association in its desire that methods employed in destroying the birds should entail the least possible suffering to them, and in consultation with the Royal Society for the Prevention of Cruelty to Animals, the Trust, with customary readiness to assist the industry, has carried out experiments on the subject at its Poultry Research Station.

The results, set out in the pamphlet, show that for large scale destruction the most effectual means is the use of petrol engine exhaust gas in a specially constructed chamber while chloroform used in a standard R.S.P.C.A. lethal chamber is particularly suitable in smaller scale operations.

RINGWORM

In our issue of August 20th, 1955, we published a note on ringworm which included lists of laboratories to which samples of suspected ringworm material could be sent. We are informed that the address given for the Bristol Hospital was insufficient, and members are asked to note that the full address is: Bristol Royal Hospital, General Hospital Branch, Guinea Street, Bristol, 1.

CONGRESS GOLF COMPETITIONS

In the men's competition for the Simpson Trophy held during the Congress week and played over the Knock Golf Club course, three of the competitors, namely Messrs. D. S. Hathorn, S. D. McAdoo and M. G. Hynes had equal points. These three competitors played off in the Whitla Hall during the President's Reception by putting nearest a mark on the dance floor. The winner of this novel play off was Mr. McAdoo, and the presentation of the cup was made to him by Mrs. Robertson.

Unfortunately, as there were too few entries for the ladies' match, the Dunkin Trophy was not competed for this year.

I.T.A. ADVERTISING ADVISORY COMMITTEE

Mr. L. Guy Anderson, D.V.M., M.R.C.V.S., of Aylesbury has been appointed to the Advertising Advisory Committee of the Independent Television Authority as a representative of the Royal College of Veterinary Surgeons and the British Veterinary Association.

CERTIFICATE FOR BRAVERY

Visitors to the Belfast Congress may have met Miss Caroline Barton Mann (17) who was presented with a cheque and a special R.S.P.C.A. certificate on Saturday, September 24th. Miss Barton Mann dived into the sea at Colwyn Bay, fully clothed, to rescue a small dog which had fallen into the water at high tide and was in danger of being dashed against the rocks.

UNIVERSITY OF LIVERPOOL

Faculty of Veterinary Science, Examination Lists, September, 1955

DEGREE OF B.V.Sc.

FINAL EXAMINATION.

Blair, Ruth, J.; Freeman, A. P.; Shepherd, P. R.

THIRD EXAMINATION.

Part II. Veterinary Pathology.

Lowe, A. D.; O'Malley, Mary C. B.; Thake, G.

Part III. Animal Husbandry.

Dunwell, R. S.; O'Malley, Mary C. B.; Thake, G.

FIRST EXAMINATION.

Adams, J.; Fitton, M. W.; Goulden, M. B.; Pickering, P. L.

NEW MEMBERS OF THE R.C.V.S.

The following graduates were admitted to membership of the Royal College of Veterinary Surgeons on the date shown against their entries:—

AKESTER, Arthur Roger, 53b, Grange Road, Cambridge. June 25, 1955 *Cambridge*. M.A., VET. M.B. (Cantab.)

GIBSON, Ian Gilbert, Briarfield, Meath Green Lane, Horley, Surrey. June 25, 1955 *Cambridge*. B.A., VET. M.B. (Cantab.)

HUNT, Michael David Nicholas, Carisbrooke, Bromley Common, Kent. June 25, 1955 *Cambridge*. VET. M.B. (Cantab.)

KENCHINGTON, Frances (Miss), Brettons, Chignall St. James, Chelmsford, Essex. June 25, 1955 *Cambridge*. VET. M.B. (Cantab).

PARSONS, Ursula (Miss), 7, Clarkson Road, Cambridge. June 25, 1955 *Cambridge*. VET. M.B. (Cantab).

SCRATTON, Edward Michael, c/o McMillan & Mott, 212, High Holborn, London, W.C.1. June 25, 1955 *Cambridge*. B.A., VET. M.B. (Cantab.)

WHITE, Geoffrey, 27, Spring Grove, Clayton West, Huddersfield, Yorks. June 25, 1955 *Cambridge*. B.A., VET. M.B. (Cantab.)

PERSONAL

Birth

RIMMER.—On September 5th, 1955, at Crosby, Liverpool, to Beryl, wife of Bernard Rimmer, M.R.C.V.S., a son, Anthony Francis.

Engagement

MALLALIEU—BRAYSHAW.—The engagement is announced between John Milne Mallalieu B.SC., M.R.C.V.S., younger son of Mr. and Mrs. S. E. Mallalieu, Ivy Cottage, Haymoor Green, Nantwich, Cheshire, and Ruth Elise Brayshaw, younger daughter of Mr. and Mrs. G. E. Brayshaw, The Croft, Masham, Yorkshire.

Marriage

GLOVER—GOLDINGHAM.—The marriage took place on September 21st, 1955, at the Church of St. Mary Magdalene, Madingley, Cambridge, of Timothy Dawson Glover, M.A., B.SC., M.R.C.V.S., of Downing College, Cambridge, and Claire Constance Goldingham of Farnham Common, Bucks.

Death

HALL.—The many friends that they made in Nigeria, Gambia and South Africa will learn with deep regret of the bereavement Dr. G. N. A. Hall sustained by the death of his wife, Nancy, at Berne in Switzerland, on August 28th, 1955. Dr. Hall, who is now in his seventies, has returned to Iraq where he is still actively at work organising laboratory services.

R.C.V.S. OBITUARY

We record with regret the death of the following member of the profession:—

HEAD, Herbert Stanley, of High House, New Romney, Kent. Graduated London, July 15th, 1909. Died September 19th, 1955, aged 71 years.

COMING EVENTS

October

1th (Thurs.). Meeting of Western Counties Division, B.V.A., at the IMPERIAL HOTEL, Exeter, 2.15 p.m. General Meeting of the Central Division, B.V.A., at the Royal Veterinary College, Camden Town, N.W.1, 5.30 p.m.

Autumn Meeting of the North of Scotland Division, B.V.A., in the Cairngorm Hotel, Aviemore, 1.30 p.m.

14th (Fri.). Annual Dinner Dance of the South Wales Division, B.V.A., at the Seabank Hotel, Porthcawl, 7.30 p.m.

20th (Thurs.). Ordinary General Meeting of the South-eastern Division, B.V.A., at the Royal Star Hotel, Maidstone, 2.30 p.m.

22nd (Sat.). General Meeting of the Society of Women Veterinary Surgeons in the Veterinary Hospital, Pembroke Gardens, Liverpool, 2.30 p.m.

26th (Wed.). Meeting of the R.A.V.C. Division, B.V.A., at the Zoological Society of London, Regent's Park, London, N.W.1, 12 noon.

29th (Sat.). Autumn Meeting of the Scottish Metropolitan Division, B.V.A., at the Scottish Milk Marketing Board, Newlands Cattle Breeding Centre, Perth, 2.45 p.m.

November

4th (Fri.). The Annual Dinner and Dance of the Royal Counties Division, B.V.A., at the Chequers Hotel, Newbury, 7.30 p.m.

R.C.V.S. EXAMINATIONS

November 4th, Friday.—Pharmacology, etc. Examination (Revised Syllabus). Written Examination.

November 7th, Monday.—Pharmacology, etc. Examination (Revised Syllabus). Oral and Practicals (London and Dublin).

November 8th, Tuesday.—Animal Management Examination, Dublin (Probable date).

December 8th, Thursday.—M.R.C.V.S. Written Examinations.

December 9th, Friday.—M.R.C.V.S. Written Examinations.

December 12th, Monday.—M.R.C.V.S. Practical and Orals commence (Panels " A " and " B ").

COUNCIL AND COMMITTEE MEETINGS IN LONDON

Wednesday, November 2nd

10.30 a.m.—Veterinary State Medicine Committee.

2.00 p.m.—Parliamentary and Public Relations Committee.

3.15 p.m.—Home Appointments Committee.

Thursday, November 3rd

10.30 a.m.—Organising Committee.

2.00 p.m.—General Purposes and Finance Committee.

Friday, November 4th

10.30 a.m.—Council.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Cumberland. Lynwood House, Popcastle, Cockermouth (Sept. 20).

Derby. Burrows Hill Farm, Brailsford (Sept. 22).

Notts. Ramsdale House Farm, Arnold, Nottingham (Sept. 21).

Salop. White House, Baschurch, Shrewsbury (Sept. 21).

Yorks. Saltmarshe Grange, Eastrington, Goole (Sept. 20).

Atrophic Rhinitis

Wilts. Donhead House Estate, Donhead St. Andrew (Sept. 22).

Fowl Pest

Lancs. Bleasdale, Marsh Lane, Longton, Preston (Sept. 22).

Norfolk. Freehold Allotments and Box Farm Allotments, Cobholm, Gt. Yarmouth (Sept. 22).

Swine Fever

Bucks. Peppers Hill Farm, Long Crendon, Aylesbury (Sept. 22).

Dorset. Tripps Farm, Purse Caundle, Sherbourne (Sept. 27).

Essex. Clock House Farm, Purleigh, Chelmsford (Sept. 21).

Lancs. Bonds Hall Farm, Elswick, Preston (Sept. 23); Laneside Farm, Downham, Clitheroe (Sept. 26).

Lincs. Roseleigh, Railway Lane, Sutton Bridge, Spalding (Sept. 27).

Notts. Whitehouse Farm, Clayworth, Retford (Sept. 22); Bon Bush Farm, Creswell, Worksop (Sept. 26).

Stirling. No. 16 Holding, Auchenreach, Milton of Campsie, Glasgow (Sept. 21); No. 15 Holding, Auchenreach, Glasgow (Sept. 22).

Sussex. Benedict House, Ewhurst, Robertsbridge (Sept. 27).

Yorks. Foston, Flaxton; 247, Batley Road, Kirkham Gate, Wakefield (Sept. 21).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

ASSISTANTS IN VETERINARY PRACTICE

Sir,—In a recent leader, the *Manchester Guardian* pointed out that the labour market has changed so much that it would now be more fitting for prospective employees to interview employers rather than vice versa. This might be worth a trial in our profession if the tactics recommended in " With Use of Car " (THE RECORD, September 10) do not meet with success. Indeed, there is really no reason why recent graduates should not advertise for employers, and older members would then realise their position *vis-à-vis* the assistants.

One visualises (in fact, one hopes to see) advertisements couched in the following terms: " Recent graduate has vacancy for wealthy practitioner. Preference given to father of nubile daughters. Good house and car essential. Apply to Box 234 enclosing testimonials from three recent employees."

I look forward to my first interview with great trepidation.

Yours faithfully,
G. B. S. HEATH.

Merrythought,
Calthwaite,
Penrith,
Cumberland.

September 19th, 1955.

DELIVERY OF "THE VETERINARY RECORD"

Sir,—It amuses me to hear so many complaints concerning the delivery of our weekly journal.

If the edition, say week ending September 10th, does not arrive until Monday, September 12th, letters of complaint stream into the Post Office and to the Editor. The whole machinery of our professional education and activity is apparently in jeopardy.

My young son of seven provided an answer to the problem last week. His space-ship comic—ante-dated "week ending 17th September"—did not arrive on Saturday, September 10th, as is usual and he was most upset. He was quite pleased, however, when it arrived on the following Monday morning, five days early.

Yours faithfully,
J. K. L. PEARSON.

Ministry of Agriculture for Northern Ireland,
Veterinary Research Division,
The Farm,
Stormont, Belfast.

September 20th, 1955.

WELLINGTON MUSEUM

Sir,—When the question was first raised as to the colour of the " Iron Duke's " famous charger Copenhagen, which is buried in the grounds at Stratfieldsaye, the future Duke (Lt.-Col. the Marquess of Douro), who is a client of mine, was away from home. I saw him to-day and he confirms that the horse was in fact a chestnut with a light chestnut mane.

Yours faithfully,
J. F. D. TUTT.

" Rothiemurchers,"
St. Cross,
Winchester.

September 21st, 1955.

ADVERTISERS' ANNOUNCEMENTS

Burroughs Wellcome & Co. announce the issue of "Temadex" brand Skin Dressing (Veterinary) for the treatment of parasitic and other skin conditions in dogs. The inclusion of three important acaricidal agents in balanced proportions, together with an antifungal substance, ensures a highly efficient and versatile skin dressing with a wide range of activity.

"Temadex," which is available to the veterinary profession only, is issued in boxes of 6 × 2 oz. tubes (Dispensing pack).

ALLEN & HANBURYS LTD. announce the introduction of Canine Distemper Vaccine (Egg Adapted-Living). This is a freeze-dried product for the immunisation of dogs against distemper and hard-pad.

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN
Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-mouth	Fowl Pest	Sheep Scab	Swine Fever
16th to 31st August, 1955		18	—	—	2	—	48
Corresponding period in	1954	11	—	—	16	—	67
	1953	12	—	—	15	—	87
	1952	71	—	9	9	—	24
1st January to 31st August, 1955		502	3	6	381	—	1,058
Corresponding period in	1954	193	3	12	591	—	1,094
	1953	477	—	28	708	—	2,121
	1952	487	—	478	151	1	644

TUBERCULOSIS (ATTESTED HERDS) SCHEMES

The number of Attested Herds, *i.e.*, herds officially certified as free from Tuberculosis as at 31st August, 195 was as follows :—

ENGLAND	WALES	SCOTLAND	TOTAL (GREAT BRITAIN)
77,190	33,359	34,893	145,442